In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 12-738V
March 1, 2013
Not to be Published

MELISSA A. HORRELL,	*	
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Petitioner,	*	
	*	
v.	*	Flu vaccine; alleged GBS five months later; motion to dismiss
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
	*	
Respondent.	*	
	*	

S. Reginald Kenan, Warsaw, NC, for petitioner.
Glenn A. MacLeod, Washington, DC, for respondent.

MILLMAN, Special Master

DECISION[1]

On October 31, 2012, petitioner filed a petition for compensation under the National Childhood Vaccine Injury Act of 1986, 42 U.S.C. § 300aa–10-34 (2006), alleging that trivalent influenza vaccine, administered October 27, 2009, caused her Guillain-Barré syndrome (GBS).

[1] Because this unpublished decision contains a reasoned explanation for the special master's action in this case, the special master intends to post this unpublished decision on the United States Court of Federal Claims' website, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (Dec. 17, 2002). Vaccine Rule 18(b) states that all decisions of the special masters will be made available to the public unless they contain trade secrets or commercial or financial information that is privileged and confidential, or medical or similar information whose disclosure would constitute a clearly unwarranted invasion of privacy. When such a decision is filed, petitioner has 14 days to identify and move to redact such information prior to the document's disclosure. If the special master, upon review, agrees that the identified material fits within the banned categories listed above, the special master shall redact such material from public access.

Prior to the first telephonic status conference in this case, the undersigned issued a 10-page Order, dated February 14, 2013, as the basis for a discussion at the first conference. The Order consists of a description of petitioner's medical records, which show that if she had GBS at all, it did not occur until five months after her vaccination. This is too long an interval to prove causation in fact from the vaccine.

On February 20, 2013, the undersigned held a Rule 4(b) Conference in this case and discussed the Order of February 14, 2013. Petitioner's counsel stated he would take the Order to his client and discuss whether or not to proceed further with the case.

On March 1, 2013, the undersigned held a telephonic status conference with the parties. Petitioner's counsel stated that his client decided not to proceed any further and moved to dismiss. The undersigned **GRANTS** petitioner's motion.

FACTS

Petitioner was born on October 31, 1967.

On October 27, 2009, she received the seasonal trivalent influenza vaccine. Med. recs. Ex. 1, at 1, 2.

On December 1, 2009, petitioner saw family nurse practitioner Sandra L. Krantz, complaining of severe pain in both legs and vomiting for one month. She had seen the psychiatrist, who kept changing her medications which made her sicker and sicker so that about two to four weeks previously, she stopped all of her medications. Since petitioner's last visit to FNP Krantz on September 23, 2009, petitioner had lost 22 pounds. She did not have diarrhea, constipation, or dysuria. She stated her legs would not support her weight. Petitioner's glucose measured 65. Med. recs. Ex. 2, at 1.

On December 30, 2009, petitioner saw FNP Krantz, complaining of leg pain, vomiting, muscle weakness, and bleeding gums. She said she could not open her mouth to fit a spoon in. Id. at 4. FNP Krantz tested petitioner's muscle strength. It was 5/5 in her upper extremities and 4/5 in her lower extremities. Her deep tendon reflexes were brisk at 2+ and equal bilaterally. Her gait was slow. She had sinus tachycardia, hypertension, and low vitamin D. Id. at 6. FNP Krantz recommended that petitioner see the neurologist Dr. Gibbs, but petitioner refused to go. Med. recs. Ex. 8, at 1.

On January 14, 2010, petitioner saw a psychiatrist. She told the doctor that she was still without medicines and had found out six days earlier from a neurologist in Greenville (presumably Dr. John W. Gibbs, III) that she has muscular dystrophy. Med. recs. Ex. 4, at 29.

On January 25, 2010, petitioner saw Dr. Robert C. Frere at East Carolina Neurology for an EMG and nerve conduction study. The results were normal. She had no evidence for lower extremity polyradiculoneuropathy, myopathy, or neuromuscular junction disorder. Med. recs. Ex. 8, at 6.

On February 10, 2010, petitioner returned to FNP Krantz complaining of leg and foot pain, severe burning pain on the top of her feet, her right leg giving way, weakness from the hips down, and her hair coming out in handfuls. Id. at 26.

On February 15, 2010, petitioner saw Dr. John W. Biggs, III, a neurologist at East Carolina Neurology, for further evaluation. Petitioner complained of a several month history of bilateral lower extremity weakness with occasional tenderness and pain in the upper extremities. Electromyography and nerve conduction studies since the last clinic visit were normal. Petitioner's blood pressure was 178/102. Her weight was 176 pounds. On physical examination,

she had normal bulk and tone in both upper extremities and lower extremities. She had 5/5 strength in her upper extremities and lower extremities. She had a normal sensory examination. Her reflexes were normal at 2+. Her gait was normal. Dr. Gibbs' diagnosis was probable fibromyalgia, significant depression, and hypertension. He prescribed Lyrica and referred her to rheumatology. Dr. Gibbs stated he did not need to see petitioner again. Id. at 3-4.

On March 16, 2010, petitioner saw FNP Krantz complaining of vomiting and painful legs and feet that had been ongoing for about five months. She has been treated for depression, hypertension, and sleep disorder. She went to New Dimensions for a psychiatric evaluation. On September 23, 2009, petitioner saw FNP Krantz with drug interactions causing severe leg pain. She stopped taking all her medications after that and her legs weakened. From September 23, 2009 to December 1, 2009, she had lost 23 pounds. She went to Greenville to see a neurologist who diagnosed her with fibromyalgia. Her pain persisted as did weight loss and vomiting. Med. recs. Ex. 2, at 29.

Also on March 16, 2010, petitioner paid another visit to the psychiatrist, who noted she does not have muscular dystrophy but instead has fibromyalgia and neuropathy. Med. recs. Ex. 4, at 27.

On March 29, 2010, petitioner went to Pender Memorial Hospital Emergency Department complaining of weakness in her legs, and numbness in her hands and face. She was diagnosed with dehydration. Id. at 10, 11, 13.

On April 3, 2010, petitioner returned to Pender Memorial Hospital ED, and was noted to be a poor historian. She complained of being weak and thirsty. By history, she had fibromyalgia, bipolar disease, and hypertension. She complained she was in pain measuring 10 out of 10. She said she had been weak for six months. She also had a deficiency in vitamins B

and D. She complained of migraines and "abdominal migraines." She was unkempt and malodorous. She complained of anxiety and depression. She was incontinent of urine and had white vaginal discharge. Petitioner spoke in a whisper. She also had tachycardia. Id. at 1, 3.

On April 3, 2010, petitioner had a CT scan of her chest, abdomen, and pelvis, showing left lower lobe consolidation most consistent with pneumonia. Id. at 8.

From April 3, 2010 to May 10, 2010, petitioner was at New Hanover Regional Medical Center. Dr. Kristi Simms did a history of present illness. Petitioner's chief complaint was vomiting. She had a significant history of psychiatric disorder. She lived independently prior to two weeks previously when she called her mother and said she was having increasing weakness and difficulty walking. She was seen at Duplin County and diagnosed with a urinary tract infection. She was treated for three days with Ciprofloxacin but had no improvement in her symptoms. About one week after completing her antibiotics, she was seen at Pender ED with the same complaint of progressive difficulty walking associated with significant nausea and vomiting which resulted in numerous choking episodes. She was diagnosed with magnesium deficiency. For the prior two days, she had been unable to eat or drink. Petitioner's mother said she had had no bowel movement for the prior two weeks. About six months previously, she had bilateral lower extremity pain and weakness. Her white blood count was 25,000. She was gastroccult positive. On chest x-ray, she had a right lower lobe infiltrate and a fluid-filled esophagus. She was diagnosed with nausea and vomiting with associated obstipation (severe constipation caused by intestinal obstruction), significant electrolyte abnormalities due to vomiting, aspiration pneumonia, multi-organ system failure secondary to sepsis, acute renal failure, acute hepatic injury, and delirium. The most likely source of petitioner's sepsis was

abdominal. She had abnormal liver function tests likely related to shock liver with dehydration. She had mixed metabolic acidosis with anion gap acidosis, and respiratory alkalosis. She also had profound weakness. Med. recs. Ex. 5, at 11-14.

On April 5, 2010, Next Wave Lab cultured petitioner's blood and found gram positive cocci in clusters and staphylococcus aureus. Med. recs. Ex. 4, at 33.

On May 10, 2010, Dr. Thomas Beckett wrote the discharge summary. Petitioner had sepsis secondary to ischemic bowel and coexisting aspiration pneumonia with respiratory failure requiring intubation and subsequent tracheostomy placement complicated by fungemia. She was admitted with sepsis secondary to mesenteric ischemia[2] and aspiration pneumonia. On April 4, 2010, she was taken to the operating room where an exploratory laparotomy, SMA (superior mesenteric artery) exploration, and ileocecectomy were performed by Dr. Eskew with preliminary findings consistent with mesenteric ischemia with ileocecal necrosis (of the junction of the small and large intestines). On April 4, 2010, at 11:00 p.m., she was taken back to the OR for a subtotal colectomy due to complete colonic necrosis. The working diagnosis was autoimmune disorder, vitamin deficiency, systemic lupus erythematosus, Sjögren's, celiac disease, HIV, Lyme disease, antiphospholipid antibody syndrome, vasculitis, heavy metal poisoning, or paraneoplastic syndrome. On April 20, 2010, Dr. Oster diagnosed petitioner with

[2] "Mesenteric artery ischemia occurs when there is a narrowing or blockage of one or more of the three mesenteric arteries, the major arteries that supply the small and large intestines." The condition is more common in people who smoke or have high blood pressure or blood cholesterol. Mesenteric ischemia may also be caused by a blood clot. These clots are more commonly seen in patients with abnormal heart rhythms. Symptoms of long-term chronic mesenteric artery ischemia are abdominal pain after eating and diarrhea. In acute mesenteric artery ischemia, symptoms are diarrhea, sudden severe abdominal pain, and vomiting. Blood tests may show a higher than normal white blood cell count in acute mesenteric ischemia and changes in the blood acid level. Mesenteric Artery Ischemia, U.S. National Library of Medicine, http://www.nlm.nih.gov/medlineplus/ency/article/001156.htm (last visited Feb. 14, 2013).

peripheral neuropathy consistent with subacute progressive sensorimotor polyneuropathy. Methotrexate and prednisone were begun for possible vasculitis. Petitioner's flaccid vocal cords were due to her underlying polyneuropathy. Petitioner was discharged to Coastal Rehab under the care of Dr. Liguori. The final diagnosis was sepsis secondary to mesenteric ischemia requiring surgery, respiratory failure with aspiration pneumonia, thrombocytopenia with resolution secondary to sepsis, malnutrition requiring a PEG tube placement, fungemia adequately treated, steroid-induced diabetes, acute renal failure, resolved, and quadriparesis with ongoing work-up of undetermined etiology also involving the vocal cords. Med. recs. Ex. 5, at 1-4.

From May 10, 2010 to August 18, 2010, petitioner was in rehabilitation. The discharge diagnosis was neurological disorder and critical illness polyneuropathy,[3] hypertension, steroid-induced diabetes mellitus resolved, acute renal failure resolved, and respiratory failure resolved. Petitioner was admitted on April 3, 2010 with sepsis secondary to aspiration pneumonia. Id. at 15.

Other doctors also opined that petitioner's neuropathy was not GBS but critical illness neuropathy. On April 16, 2010, Dr. David M. Schultz, a neurologist, did a physical examination of petitioner, finding she had motor strength of 2/5 throughout, dysesthesias in all four

[3] "Critical illness neuropathy is a disease of peripheral nerves, occurring as a complication of severe trauma or infection (critical illness). It develops while patients are in the intensive care unit and it is typically diagnosed by limb weakness and unexplained difficulty in weaning from mechanical ventilation." http://www.aanem.org/education/patient-resources/disorders/critical-illness-neuropathy.aspx. Articles describing critical illness neuropathy are "Clinical Review: Critical illness polyneuropathy and myopathy" by G. Hermans, et al., 12 Critical Care 238 (Nov. 25, 2008), http://ccforum.com/content/12/6/238, and "Clinical Outcomes of Critical Illness Polyneuropathy" 22 Pharmacotherapy 3 (2002), http://www.medscape.com/viewarticle/432050 (the authors surmise that critical illness polyneuropathy occurs in 70% of patients with sepsis).

extremities, and absent deep tendon reflexes. He opined that petitioner had possible neuropathy, multifactorial. The etiology could be critical illness neuropathy vs. peripheral neuropathy. Id. at 20-21. He wrote, "Given the length of time of weakness, this is less likely to be Gullain-Barre syndrome." Id. at 22. On April 23, 2010, Dr. Richard F. Trotta noted, "While Guillain Barre syndrome and its variance are possible, they appear less likely." Id. at 29, 32.

On May 6, 2010, Dr. Roger McLendon had an addendum to a report on a sural nerve biopsy, in which he states, "Given the differential diagnosis, there is no obvious evidence of active Guillain-Barre syndrome. The differential diagnosis includes critical care polyneuropathy; however, a recovering GBS cannot be ruled out although there is no obvious sprouting suggestive of recovering phase." Med. recs. Ex. 7, at 4. On May 13, 2010, Dr. McLendon wrote another addendum to the report of the sural nerve biopsy, which showed moderate to severe axonal degeneration without regeneration. In this addendum, he stated, "The multifocal collections of inflammatory cells are most likely macrophages responding to injury. The absence of a significant perivascular lymphoid population makes an inflammatory neuropathy less likely." Med. recs. Ex. 5, at 56.

On September 23, 2010, Dr. Anne F. Buckley interpreted a left gastrocnemius muscle biopsy. Her findings were "prominent active myopathic process with evidence of chronicity. The chronic features indicate that the myopathy precedes the patient's episode of critical illness, and may be the reason for her several months of weakness prior to her acute illness. The pattern of damage is suggestive of small-vessel ischemia. Dermatomyositis is therefore in the differential diagnosis, as this disorder can be associated with bowel ischemia." Id. at 58. Other possibilities included autoimmune disorders or a subacute infectious endocarditis. Id.

DISCUSSION

Petitioner's unfortunate medical problems do not include GBS in close proximity to her vaccination. Regardless of the statements of various doctors which petitioner filed--Dr. Liguouri (flu vaccine could have caused petitioner's GBS), Dr. Gernelli, and Dr. Sessions (flu vaccine could have caused petitioner's GBS) (see Exhibit 6, at 1, 10, and Exhibit 9, at 1)--petitioner did not have a neurologic illness much less GBS before April 2010 because she tested in January and February 2010 as neurologically normal. These doctors appear not to have read Dr. Frere's electromyographic and nerve conduction results on January 25, 2010 that petitioner did not have any evidence of neuropathy. These doctors also appear not to have read Dr. Gibbs's February 15, 2010 analysis that petitioner was normal neurologically after examining her and finding she had normal bulk, tone, reflexes, strength, gait, and sensation. Someone with normal nerve conduction, strength, gait, reflexes, and sensation does not have GBS.

Even assuming petitioner had GBS and not critical illness neuropathy in April 2010, the onset interval between her influenza vaccination and her neuropathy was five months. The undersigned has never held that vaccination causes GBS or any other demyelinating illness after a two-month interval between vaccination and onset. See Corder v. Secretary of Health & Human Services, No. 08-228, 2011 WL 2469736 (Fed. Cl. Spec. Mstr. May 31, 2011), in which the undersigned dismissed a case in which petitioner alleged that influenza vaccine caused her GBS four months later. The reason for the dismissal was that the onset interval was too long.

To satisfy her burden of proving causation in fact, petitioner must prove by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen v. Sec'y

of HHS, 418 F.3d 1274, 1278 (Fed. Cir. 2005). In Althen, the Federal Circuit quoted its opinion in Grant v. Sec'y of HHS, 956 F.2d 1144, 1148 (Fed. Cir. 1992):

> A persuasive medical theory is demonstrated by "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury[,]" the logical sequence being supported by "reputable medical or scientific explanation[,]" i.e., "evidence in the form of scientific studies or expert medical testimony[.]"

Without more, "evidence showing an absence of other causes does not meet petitioners' affirmative duty to show actual or legal causation." Grant, 956 F.2d at 1149. Mere temporal association is not sufficient to prove causation in fact. Id. at 1148.

Petitioner must show not only that but for flu vaccine, she would not have GBS (if she indeed had it), but also that the vaccine was a substantial factor in causing her GBS. Shyface v. Sec'y of HHS, 165 F.3d 1344, 1352 (Fed. Cir. 1999). This she has not done.

Petitioner has failed to make a prima facie case of causation in fact, and, recognizing this, has moved for dismissal. The undersigned grants her motion. This petition is hereby **DISMISSED**.

CONCLUSION

Petitioner's petition is **DISMISSED**. In the absence of a motion for review filed pursuant to RCFC Appendix B, the clerk of the court is directed to enter judgment herewith.[4]

IT IS SO ORDERED.

Dated: March 1, 2013

s/Laura D. Millman
Laura D. Millman
Special Master

[4] Pursuant to Vaccine Rule 11(a), entry of judgment can be expedited by each party's filing a notice renouncing the right to seek review.